Learning objective

Understand that amiodarone pulmonary toxicity can occur acutely and identify the risk factors for it.

CASE

A 72-year-old male with diabetes mellitus, hypertension, paroxysmal atrial fibrillation presented to the hospital due to recurrent falls, fatigue and generalized muscle aches. He was initially admitted for a heart failure exacerbation. Through the hospitalization, he developed lumbar back pain leading to an MRI of his lumbar spine. This imaging showed abnormal marrow signal in the right posterior elements of L3-L4 with soft tissue collection and edema, suspicious for osteomyelitis of the spine. He received broad-spectrum antibiotics. He then underwent L2-S1 bilateral decompressive laminectomies with removal of a large epidural abscess. Postoperatively, he had multiple episodes of rapid atrial fibrillation and was started on a diltiazem drip. This resulted in persistent hypotension and he was switched to an amiodarone drip. He was on the amiodarone drip for three days with better control of his heart rate. He was then switched back to his home dose of diltiazem. He started to develop respiratory failure, with rapidly increasing oxygen requirement. Chest radiography was notable for increasing opacities. Aggressive diuresis did not improve his respiratory status. He was then intubated and underwent bronchoscopy and bronchoalveolar lavage (BAL). Flow cytometry of the BAL sample showed lymphocytes, predominantly T cells. This finding was concerning for drug toxicity, likely amiodarone. He was then treated with high dose steroids and was quickly extubated.

Amiodarone is an antiarrhythmic agent that is frequently used in supraventricular and ventricular arrhythmia. It has been associated with toxicity due to its accumulation in different organs, including thyroid, lungs and skin. The risk of amiodarone pulmonary toxicity (APT) increases with higher plasma concentration of the drug and hence correlates with total cumulative dose. However, this toxicity can also occur acutely, and is sometimes seen in patients postoperatively, despite a short course. Amiodarone-induced ARDS is also a rare complication in postoperative patients. High oxygen concentrations increase the risk for the development of amiodarone pulmonary toxicity. In some studies, the mortality of amiodarone-induced ARDS after surgery was as high as 10%. Therapy includes discontinuation of the drug and corticosteroids.

APT is a known complication of long-term use of this amiodarone; however, clinicians need to be aware that this toxicity can occur with a short course of the drug as well. Conditions that predispose patients to acute APT was postoperative stage and prolonged intubation. Therapy includes prompt discontinuation of the drug, and hence physicians need to have a high suspicion to prevent worsening respiratory status in these patients.

References: